new in our experience, was the lovely flowers furnished by the Women's Entertainment Committee, under the chairmanship of Mrs. David B. Ludwig, for all occasions and changes of floral decorations for all public meetings.

DENTAL FLUOROSIS AND DENTAL CARIES

FOR some 8 or 9 years past the presence of fluorine in water supplies has been attracting much attention, chiefly as a danger and as the cause of endemic dental fluorosis, or mottled enamel, which has been reported in some 300 communities in 23 states of the Union.

According to Dean,¹ this condition was first described by J. M. Eager ² in a report from Naples under the name "Denti di Chiaie (Chiaie teeth)," so called from Dr. Stephano Chiaie, who first observed the condition in Italy, where it was ascribed to volcanic fumes in the air or dissolved in drinking water. Italian immigrants not infrequently consulted doctors in this country for the condition, which was "unlike any other dental disease."

Apparently nothing was done until 1916, when Black and McKay^{3, 4} published a series of articles on the subject, Black describing it as "An Endemic Developmental Imperfection of the Teeth, Heretofore Unknown in the Literature of Dentistry." From that time until about 1932, the condition does not seem to have attracted much general attention. However, Black observed at that time some apparent relation between mottled enamel and dental caries. Other observations along this line were not abundant, but intensive work has been going on for 5 or 6 years, notably in the National Institute of Health and in some western states.⁵

The condition has been proved to be due to the use of water carrying toxic amounts of fluorides during the period of dental calcification. However, evidence has accumulated which indicates that a certain amount of fluorine in the water exercises some inhibitive action against caries, or, at least, that there is an inverse relationship between endemic dental fluorosis and dental caries. Many factors are involved but the careful plan followed in the study discussed here seems to have eliminated as many as possible of these.⁶

The four cities of Macomb, Quincy, Galesburg, and Monmouth, located near together in the flat country of North Central Illinois, were selected for comparison, and the examinations were limited to white children 12, 13, and 14 years old. The population in these four towns is very similar and they have approximately the same percentage of native white inhabitants—88–97. The location insures comparatively equal sunlight intensity.

In Galesburg and Monmouth the water supply contains from 1.7 to 1.8 parts per million of fluorides, whereas that of Quincy and Macomb has only 0.2 parts per million. Of 319 children in Galesburg and 148 in Monmouth, the number of carious permanent teeth per 100 children was 201 and 205 respectively, whereas of 112 children in Macomb and 306 in Quincy, the carious teeth were 401 and 633 respectively. There was twice as much dental caries in Macomb and more than three times as much in Quincy, where the water was practically free from fluorides, as at Galesburg or Monmouth. About 35 per cent of children examined in Galesburg and Monmouth were free from caries, while in Macomb and Quincy only 14 and 4 per cent respectively were free.

Another test was observation of interproximal, or smooth surface, caries for which the four superior anterior teeth of children who had used the city water supplies continuously throughout their lives were selected. In Galesburg and Monmouth, 2,718 surfaces examined showed an average of 0.59 carious lesions per 100 surfaces, whereas of 2,814 surfaces of children in Quincy and Macomb, there were 8.9 carious lesions per 100 surfaces, or 16 times as much.

An unusual feature of this investigation concerns the presence of Lactobacillus acido philus which is believed by many to be a dependable index of the dental caries activity. Such examinations were made only at Quincy and Galesburg, where caries in permanent teeth amounted to 636 per 100 children in Quincy, 3.4 times the amount found in Galesburg, 189 per 100. The percentage of all acidophilus counts of 30,000 or over is also 3.4 times as high in Quincy as in Galesburg, possibly a coincidence, but an interesting finding in this general study.

This report, which does not pretend to be final, holds that from the epidemiological standpoint the difference in the water supplies must be considered the cause of the differences in the dental condition of the children examined. This points to the possibility of at least partial control of dental caries through the public water supplies. All studies of dental caries should be accompanied by complete chemical analyses of the domestic waters used, especially with regard to the comparatively rare elements.

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THE TOXICITY OF SELENIUM

I N a recent article 1 attention was called to the problem of selenium poisoning, the danger from which seems to be increasing through the use of spraying, irrigation, and certain industrial practices. While we know little of selenium poisoning in human beings, the deleterious effects on livestock and poultry have been proved.

The question has been under study at the National Institute of Health for Their latest report 2 is of great interest as showing the relation between the composition of the diet and the danger of poisoning. In all of their experiments a prominent characteristic of selenium poisoning has been in evidence; namely, the great variation in individual as well as in species susceptibility. Many experiments have been made in trying to find the cause of this. It now seems certain that as far as rats go the toxicity of selenium taken in food is largely determined by dietary factors. A quantity of selenium which proves highly toxic and damaging to the tissues when given in a diet low in protein and high in carbohydrate is only slightly or not at all harmful when fed in another diet high in protein and law in carbohydrate. In other words, all the available evidence indicates that the toxicity of naturally occurring food selenium is determined to a great extent not so much by the level of intake of selenium, as by the ratio between protein and selenium in the diet. While 10 parts per million of selenium, a ratio of 1 per cent protein in the diet to about 30 micrograms of